

# FORAGING ECOLOGY AND DEPREDACTION MANAGEMENT OF GREAT BLUE HERONS AT MISSISSIPPI CATFISH FARMS

JAMES F. GLAHN, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, National Wildlife Research Center, P.O. Drawer 6099, Mississippi State, MS 39762, USA

BRIAN DORR, <sup>1</sup> U.S. Department of Agriculture, Animal and Plant Health Inspection Service, National Wildlife Research Center, P.O. Drawer 6099, Mississippi State, MS 39762, USA

J. BRENT HARREL, <sup>2</sup> U.S. Department of Agriculture, Animal and Plant Health Inspection Service, National Wildlife Research Center, P.O. Drawer 6099, Mississippi State, MS 39762, USA

LESTER KHOO, College of Veterinary Medicine, Mississippi State University, P.O. Box 197, Stoneville, MS 38776, USA

**Abstract:** Great blue herons (*Ardea herodias*) occur at high densities at catfish farms in the southern United States. They are perceived by farmers to prey heavily on fish stocks. After a field study at selected catfish farms in Mississippi, we describe (1) the pond conditions where great blue herons intensively foraged, (2) the prevalence of disease in catfish that were captured by herons, and (3) predation rates and economic effects of herons among selected pond situations. Heron abundance was significantly associated with season and was greatest during the fall (Sep–Oct). We characterized catfish ponds as having high ( $\geq 6$  birds) or low ( $\leq 3$  birds) foraging activity by herons and characterized pond type, disease prevalence, and water quality. Categorical models showed a significant association of heron activity with disease prevalence in ponds (diseased) and fingerling ponds. Based on model parameters and associated odds ratios, high heron activity was 6.6 times greater at fingerling ponds than at food-fish ponds, and 40.1 times greater at diseased ponds than at those without diseased fish. This was presumably because fingerlings are a more desirable prey size, and disease makes catfish more vulnerable to heron predation. Based on pathology reports, 85% ( $n = 55$ ) of the live catfish captured by herons from high-activity ponds were diseased, of which 76% were considered to have a terminal condition. In contrast, 75% ( $n = 63$ ) of the catfish captured by herons congregated at ponds where catfish were being fed were diagnosed as healthy and only 3 (5%) were considered to have a terminal condition. Although both disease and fish feeding bring catfish to the surface and increase their vulnerability to heron predation, we suggest that heron harassment efforts by farmers be focused during fish feeding when heron capture rates are the highest and the greatest predation on healthy catfish occurs. Based on heron foraging rates, average numbers of herons seen, and the duration of foraging activity, we estimated low expected heron predation losses at catfish ponds over time. Assuming that predation losses observed in this study are representative, we conclude that catfish predation losses from great blue herons are either insignificant or readily preventable.

**JOURNAL OF WILDLIFE MANAGEMENT 66(1):194–201**

**Key words:** *Ardea herodias*, channel catfish farms, flocking behavior, foraging, great blue heron, *Ictalurus punctatus*, Mississippi, predation.

Great blue herons (*Ardea herodias*) frequently are implicated in depredation problems at aquaculture facilities throughout the United States (Hoy et al. 1989; Parkhurst et al. 1992; Stickley et al. 1995; Pitt and Conover 1996; Glahn et al. 1999a,b). Most depredation problems have been documented at trout-rearing facilities (Parkhurst et al. 1992; Pitt and Conover 1996; Glahn et al. 1999a,c), but studies of heron predation on commercial channel catfish (*Ictalurus punctatus*) have shown mixed results. In a survey of catfish producers, the great blue heron was cited by 42% of the respondents as causing depredations on their fish stocks (Wywiałowski 1999). The only bird spe-

cies cited more often (53%) was the double-crested cormorant (*Phalacrocorax auritus*).

Two field studies have indicated a potential for great blue herons to cause significant economic losses, with replacement costs for catfish removal ranging from US\$3,800 to US\$11,400 per year for the average catfish farm (Stickley et al. 1995, Glahn et al. 1999b). These studies revealed that <50% of the heron diet was live catfish and that most catfish are lost to heron predation during the spring and fall when the incidence of catfish disease is highest. Thus, realized economic losses attributable to herons must account for the number of catfish that would have died from disease.

Studies with captive herons foraging on catfish have helped elucidate questions about realized losses. Dorr et al. (1998) found that captive herons could maintain their body mass at simulated catfish ponds only during episodes of fish disease or

<sup>1</sup> E-mail: brian.s.dorr@usda.gov

<sup>2</sup> Present address: U.S. Fish and Wildlife Service, Kentucky Field Office, 3761 Georgetown Road, Frankfort, KY 40601, USA.



where supplemental food was available. Herons lost body mass when only healthy catfish were available. Glahn et al. (2000) confirmed the results of Dorr et al. (1998) and found no evidence of significant production losses caused by heron predation. Together these results suggest that herons primarily prey on diseased catfish and are unlikely to impact catfish production. However, additional studies were needed to substantiate these findings under actual field conditions and to clarify the extent to which herons prey on healthy catfish brought to the surface during fish feeding and conditions of low dissolved-oxygen levels.

We undertook this study to address some remaining questions concerning great blue heron predation at catfish farms. We studied heron activity among catfish ponds and its relationship to the prevalence of disease, water quality, pond type, fish feeding, and seasonality. The objectives of this study were to (1) determine whether herons selectively forage at specified catfish pond types (food-fish and fingerling ponds) or under specific conditions (disease, poor water quality); (2) determine the prevalence of disease in catfish that are captured by herons; and (3) compare foraging rates and relative economic effects of herons among selected pond situations where predation is observed to occur.

## STUDY AREA AND METHODS

### Study Design and Heron Censuses

We selected 4 catfish farms in the Mississippi Delta region based on producer reports of moderate to severe heron predation. Two farms were selected in the eastern delta and 2 farms in the western delta. Farms ranged in size from 76 ha to 2,145 ha. Within each farm, we selected 1 to 7 blocks of contiguous ponds, with the number of blocks being proportional to farm size (i.e., the entire 76-ha farm constituted 1 block, and a portion of the 2,145-ha farm contained 7 blocks). We selected blocks from within farms where the most herons were seen during preliminary censuses. We selected 16 blocks. Eight blocks were selected from farms 1 and 2 in the eastern delta (1 and 7 blocks, respectively), and the remaining 8 blocks from farms 3 and 4 in the western delta (4 blocks each). Each of the 16 total blocks contained from 10 to 34 ponds ( $n = 314$ ), with ponds averaging about 6 ha (i.e., blocks contained between 60 and 204 ha of ponds).

Initially, we categorized ponds as food-fish ponds (Food), which contained both small (<20 cm) and large (>30 cm) fish in a multi-batch cropping sys-

tem (Tucker and Robinson 1990); fingerling ponds (Fing), which contained only small fish up to 20 cm in length; and brood-fish ponds (Brood), which contained primarily large (>40 cm) fish used for breeding purposes. Because Brood ponds were too few in number to be analyzed separately, we combined them with Food ponds because of their similarity in fish type. For each block, we established a census route along the pond levee roads. This allowed us to census herons by using a vehicle that also doubled as a blind.

We censused each block once or twice each month from July to December 1998. We selected this period because it coincided with the highest densities of herons on catfish farms (Glahn et al. 1999b). We initiated censuses either 1 hr after sunrise or 2 hr before sunset to coincide with foraging activity of herons (Ross 1994, Glahn et al. 1999b). We assigned censuses to 1 of 3 seasons: summer (Jul–Aug), fall (Sep–Oct), and winter (Nov–Dec). We used a fixed effects, repeated measures generalized linear model to detect differences among censuses (PROC GENMOD; SAS Institute 1996). The between-measures factor was pond type (2 levels) and the within- (repeated) measures factor was season (3 levels). Because counts of herons fit a Poisson distribution better than a normal distribution, we specified the model to assume a Poisson distribution. We used a chi-square test to test for model effects.

### Pond Observations

We conducted detailed observations of heron foraging activity immediately after each census and during periods of fish feeding. Based on the census, we assigned ponds to 1 of 2 categories: (1) ponds with high heron activity (HHA) were defined as having at least a 3-fold difference between the minimum and maximum number seen in a given block, or a minimum of 6 herons; or (2) ponds with low heron activity (LHA) were defined as having 3 or fewer herons. A separate subgroup of HHA ponds was made up of those where herons congregated when fish had just been fed floating feed (i.e., had fish feeding activity [FFA]). All pond observations were conducted in an identical manner, except that FFA observations focused only on the period when catfish came to the surface to obtain floating feed.

We drove to a position within 50 to 100 m from water's edge where the entire pond could be observed, typically 250 to 400 m from the nearest bird, and counted the number of herons present on the pond. If all the herons had flushed from

the pond, the observer waited up to 20 min for 1 or more herons to return before starting the observation. Using the vehicle as a blind (Pitt and Conover 1996), we watched the entire pond edge for 1 hr with binoculars and recorded the following behaviors: (1) the total number of herons entering and leaving the pond edge at 1-min intervals over the observation period, and (2) the total number of live and dead catfish captured by all birds. We judged whether a catfish was alive by its movement in the bill of a heron.

We pooled data by farm to obtain a sufficient sample of observations for each pond type (HHA, LHA, and FFA) for analyses. We calculated the bird-minutes of heron activity for each observation period by summing the number of herons present over time, disregarding heron numbers at the start and the end of the observation. Foraging rates among farms and pond types were calculated by dividing the total number of catfish consumed by the total number of bird-min of activity per farm and activity type. We used a 1-way analysis of variance (ANOVA) and a Tukey's Studentized Range Test (PROC GLM; SAS Institute 1994) to examine differences among mean heron foraging rates for HHA, LHA, and FFA ponds from randomly distributed data collected over the course of the study.

### Health Status of Live Catfish Captured by Herons

During 1998, we used 2 methods to collect catfish from herons seen capturing live catfish at HHA, LHA, and FFA ponds. We collected fish from herons shot with a .22-250 caliber rifle, using hollow-point ammunition or from herons that dropped their fish after being fired upon. We focused on live catfish captured because we did not consider consumption of dead catfish to be of economic importance. We supplemented heron collections during the summer of 1999 on FFA ponds to increase our sample of catfish taken under these situations. We also attempted to sample herons on LHA ponds for both years, but the low numbers of herons foraging and their low capture rates of live catfish precluded us from including this pond type in our analysis.

Immediately after each heron collection, we removed the stomach and esophagus and removed and measured (to the nearest millimeter) all intact and partially intact catfish. The total length of partially intact catfish was derived from a regression equation that related the total length to the distance between the base of the adipose

fin and the distal end of the caudal fin (Glahn *et al.* 1998).

Immediately following measurement, we placed intact catfish in doubled plastic freezer bags and put them on ice in an insulated ice chest. Intact catfish were transported to the laboratory within 24 hr after collection, at which time L. Khoo assessed the health status of the fish in single blind fashion (by including healthy fish in submitted samples). We performed routine diagnostic procedures on all fish to identify any manifestation and severity of disease (Plumb and Bowser 1983). We performed gross examinations for external and internal lesions. Microscopic examination (wet mounts) of gill clips and, where present, skin lesions provided diagnostic evidence of disease and parasitism.

Following necropsy, we collected portions of the gill, spleen, heart, brain, stomach, intestine, liver, and kidney from each fish and placed them in neutral buffered 10% formalin. To confirm the diagnosis and severity of infection, we processed these tissues using routine histological techniques. We stained sectioned mounts with hematoxylin and eosin and examined the slides via light microscopy. We also performed bacterial cultures of the brain and posterior kidneys to confirm the causal agent of disease. In addition, we performed virus isolation procedures to confirm Channel Catfish Virus (CCV) when we found gross lesions on catfish fingerlings during the summer months.

Bacterial cultures utilized trypticase soy agar (TSA) with 5% defibrinated sheep blood as well as dilute Mueller Hinton agar. We incubated bacterial cultures at 25 °C and examined them daily for 96 hr to identify Enteric Septicemia of Catfish (ESC) and columnaris, the predominant bacterial diseases infecting cultured channel catfish (Tucker and Robinson 1990).

For virus isolation we titrated portions of the spleen, posterior kidney, and anterior kidney together with 5 ml of sterile Hank's buffered salt solution and centrifuged the suspension at approximately 1000× G for 15 min at room temperature (22 °C). The supernatant was filter-sterilized using a 0.45-μm syringe filter (Acrodisc; Gelman Sciences, Ann Arbor, Michigan, USA). A 0.5-ml aliquot of the supernatant was used to inoculate a confluent culture of channel catfish ovary cells (Bowser and Plumb 1980). We incubated these cultures at 25 °C for 7 days and made daily observations for cytopathic effects associated with CCV.

We identified fish diseases and parasites, if present, and we categorized the condition of the fish as: healthy—no pathological sign of disease was

observed; mildly infected—clear pathological signs of disease, but the fate of these fish could not be determined; terminal infection—fish in an advanced stage of disease that precluded their survival; or unknown—damage to the specimen precluded ruling out infectious agents.

### Pond Condition Assessment

We compared heron activity for paired HHA and LHA ponds as a function of pond type and water quality conditions affecting fish health. We excluded FAA ponds from this analysis because the event causing heron activity (i.e., fish feeding) was already known. Where more than 1 LHA pond was associated with a HHA pond within a block, 1 LHA pond was randomly selected for comparative analysis. We used a categorical model (PROC GENMOD; SAS Institute 1996) with a logistic, dichotomous response variable in which the response variable entered the model as either a 1 for HHA or a 0 for LHA. Explanatory variables used in the model included pond type (Food or Fing), prevalence of disease (Yes or No), dissolved oxygen (Low or Norm) and water chemistry (Poor or Norm), and a pond type  $\times$  disease interaction term. We used a chi-square test to determine significance of the explanatory variables. We used parameter estimates of significant explanatory variables ( $P \leq 0.05$ ) to calculate the odds ratio of HHA relative to LHA using the formula,  $\text{odds} = e^{B_i}$ , where  $B_i$  = the parameter estimate of the explanatory variable (SAS Institute 1996). Because the data were binomial, we specified that the model should assume a binomial distribution.

We obtained the categorical data on paired ponds by 2 methods, empirical measurements and farm-manager surveys. Empirical measurements taken by us included the abundance of dead and dying fish on the pond edge (as an index of disease prevalence), dissolved oxygen, temperature readings, and water samples to determine water chemistry. We interviewed farm managers about their records concerning pre-dawn dissolved-oxygen levels and fish diseases known to be present in each of the paired ponds.

To estimate the abundance of dead and dying fish, we located the leeward corner of the pond where dead fish, if present, were most concentrated. From this corner, we located a starting point for sampling by pacing 10 m away from the corner down the short side of a rectangular pond. From this starting point, we placed a 1-m hoop onto the pond edge and counted all dead and dying fish or portions of fish encircled by the

hoop. We collected data from 30 hoop samples spaced at 3-m intervals in the vicinity of the leeward corner of each pond sampled.

At the leeward corner of the pond and the corner diagonally across from it, we measured dissolved oxygen and temperature with a calibrated meter YSI model 55 (Tucker and Robinson 1990). At each of the same corners, we collected a 50-ml water sample for analysis of water chemistry, including alkalinity, chlorides, ammonia, nitrates, nitrites, and pH (Tucker and Robinson 1990).

We considered a pond to be diseased if the farm manager could identify the pond as diseased. If the manager was unavailable or could not identify disease in a specific pond, we categorized that pond as diseased if the mean number of dead and dying fish in hoop samples exceeded the mean for all ponds categorized as diseased. We considered dissolved-oxygen levels to be low if pre-dawn manager records or our readings were below 3 ppm. Water chemistry was judged poor if the chloride:nitrite ratio was  $<10$ .

### RESULTS

Great blue heron activity on catfish ponds was highly clustered on relatively few ponds. Most ponds (68.5%) had no heron activity, while high heron activity ( $\geq 6$  birds) occurred on only 6.6% of the ponds. Despite their infrequent occurrence, HHA ponds accounted for 60.2% of the total number of herons counted. Overall, mean heron numbers during pond observations were relatively low (1–2 birds/pond). Herons were most abundant during the fall (Sep–Oct) and winter (Nov–Dec) months ( $\chi^2 = 22.49$ ,  $P < 0.001$ ; Fig. 1).

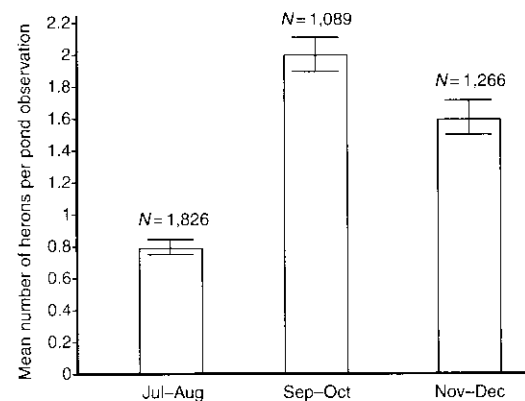


Fig. 1. Bimonthly changes in the mean number of herons observed per pond observation during repeated censuses of 314 catfish ponds from July through December 1998 in the Mississippi delta region.

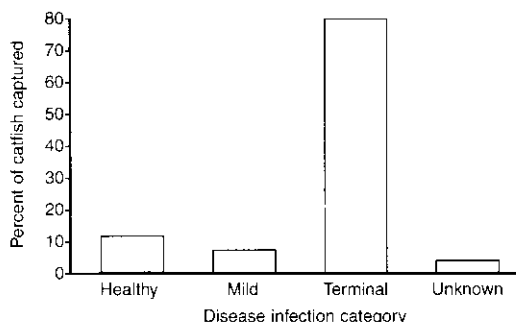


Fig. 2. Percentage of catfish ( $n = 55$ ) clinically diagnosed by disease category that were taken by great blue herons at selected catfish ponds in the Mississippi delta region where herons were concentrated ( $\geq 6$  birds) at these ponds from July through December 1998.

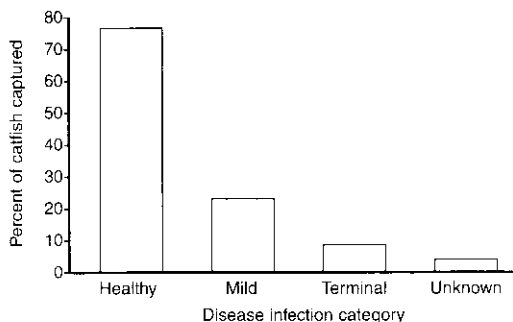


Fig. 3. Percentage of catfish ( $n = 63$ ) clinically diagnosed by disease category that were taken by great blue herons at selected catfish ponds in the Mississippi delta region where catfish had been recently fed floating feed during the summers (Jul through Sep) of 1998 and 1999.

Herons were concentrated on HHA ponds and at catfish ponds during fish feeding ( $\bar{x} = 6.86$ ,  $n = 28$ ,  $SE = 0.90$  and  $\bar{x} = 4.5$ ,  $n = 39$ ,  $SE = 0.65$ , respectively). Results from the Tukey's Studentized Range Test indicated that among the 4 farms studied, herons had higher ( $P \leq 0.05$ ) capture rates on live catfish at FFA ponds ( $\bar{x} = 0.0252$  live catfish/bird-min,  $n = 4$ ,  $SE = 0.0089$ ) than on either HHA ponds ( $\bar{x} = 0.0036$  live catfish/bird-min,  $n = 4$ ,  $SE = 0.0011$ ) or LHA ponds ( $\bar{x} = 0.0015$  live catfish/bird-min,  $n = 4$ ,  $SE = 0.0005$ ). Despite higher capture rates of live catfish during FFA events, their limited duration ( $\bar{x} = 28.2$  min,  $n = 26$ ,  $SE = 2.28$ ), relative to events such as disease outbreak, limits exposure of catfish to predation. We saw no dead catfish consumed during FFA events. However, capture rates of live and dead catfish at HHA ponds ( $\bar{x} = 0.0045$  dead catfish/bird-min,  $n = 4$ ,  $SE = 0.0020$ ) were similar. This similarity in capture rates for live and dead catfish was also observed for LHA ponds ( $\bar{x} = 0.0010$  dead catfish/bird-min,  $n = 4$ ,  $SE = 0.0008$ ).

The categorical comparison of the characteristics for HHA and LHA ponds indicated no interaction of pond type and disease prevalence ( $\chi^2 = 1.31$ ,  $P = 0.25$ ). However, pond type and disease prevalence were important in explaining variation in heron activity ( $\chi^2 = 11.46$ ,  $P = 0.0007$  and  $\chi^2 = 40.76$ ,  $P < 0.0001$ , respectively). Considering pond type, the model for type Fing provided a significant ( $\chi^2 = 9.34$ ,  $P = 0.0022$ ) parameter estimate of 1.8845 ( $SE = 0.6166$ ). Considering the prevalence of disease, diseased ponds provided a significant ( $\chi^2 = 23.38$ ,  $P = 0.0001$ ) parameter estimate of 3.6907 ( $SE = 0.7633$ ). Odds ratios for these parameter estimates indicate that HHA

ponds were 6.6 times more likely to occur on fingerling ponds than food-fish ponds and 40.1 times greater on diseased ponds than healthy ponds. Other pond characteristics occurred too infrequently to be considered in the model. Only 4 of 87 (4.6%) dissolved-oxygen measurements were below normal ( $< 3$  ppm), and there was only 1 instance where water chemistry was judged as poor (ratio of chloride:nitrite  $< 10$ ).

Eighty-five percent ( $n = 55$ ) of the live catfish captured by herons from HHA ponds were diseased, and 76% were considered to have a terminal infection (Fig. 2). In contrast, 74.6% of the 63 catfish obtained from FFA ponds were diagnosed as healthy, and only 5% of these cases were diagnosed as having a terminal infection (Fig. 3). Catfish specimens obtained from those captured but not consumed by herons had a mean total length of 220 mm ( $n = 108$ ,  $SE = 7.77$ ). However, lengths of catfish in the stomach contents of herons collected were smaller ( $\bar{x} = 147.9$  mm,  $t = 5.94$ ,  $P < 0.0001$ ).

## DISCUSSION

The distribution of great blue herons at catfish farms rarely has been studied. Such investigations provide important clues to where fish prey may be most available. During our study, herons concentrated on ponds stocked with fingerlings, diseased catfish ponds, and ponds where floating feed had been recently fed to catfish. This is consistent with studies suggesting that captive herons feed opportunistically on fish available near the surface of the water and that herons are inefficient at capturing healthy catfish (Dorr et al. 1998, Glahn et al. 2000). Fingerling ponds were 6.6 times more likely to be classified as having high

heron activity relative to food-fish ponds and 40.1 times more likely to be classified as having high heron activity for diseased ponds versus healthy ponds. Hodges (1989), who censused heron numbers on Mississippi Delta catfish farms from October to April, reported that during 1 survey 92% of 87 herons were distributed on 2 ponds where a shad (*Dorosoma* sp.) die-off was occurring. During our study, most of the herons also were concentrated on relatively few ponds. Consistent with previous studies of heron foraging on catfish ponds (Stickley et al. 1995, Glahn et al. 1999b), a higher percentage of these concentrations occurred during the fall when catfish diseases are more prevalent (Tucker and Robinson 1990).

The high concentrations of herons foraging on diseased ponds probably have negligible economic effects on catfish production because most live catfish consumed were terminally infected with disease. Therefore, these fish would be unlikely to contribute to the market value of the standing crop at harvest. This view of losses assumes that heron foraging activity is not responsible for promoting fish diseases that are ubiquitous in cultured catfish populations (Waterstrat et al. 1999) or for causing injury to healthy catfish not captured (Parkhurst et al. 1992).

The extent of losses of healthy catfish during fish feeding would be significant if it were not limited by the short duration of the feeding events and the limited nature of catfish feeding throughout the year. During fish feeding, we observed an average of 4.5 herons foraging for approximately 28 min and obtaining 0.025 fish/heron-min. This would amount to a total take of 3.1 catfish per fish-feeding event. Considering that ponds are typically fed once per day during half of the year from 15 April to 15 October (Lovell 1989), the annual loss per pond would be only 575 fish, or 0.7% of fish stocks in food-fish ponds. In fingerling ponds, the percent loss would be considerably less because stocking rates are >10 times those of food-fish ponds.

Low-heron-activity ponds realize lower potential economic losses. On our LHA ponds, we found a mean of 0.57 birds over the study period. At a foraging rate of 1.3 fish consumed per day, the annual loss would be estimated at 282 catfish or only 0.38% of the minimum of 75,000 fish in a typical food fish production pond.

Capture rates of herons on catfish have been previously reported (Stickley et al. 1995, Glahn et al. 1999b) to range from 0.004 to 0.013 catfish/bird-min. In contrast, herons preying on trout in

clear-water raceway situations have predation rates ranging from 0.033 to 0.05 trout/bird-min (Pitt and Conover 1996, Glahn et al. 1999a). However, events that bring catfish close to the surface can increase their availability to herons, as evidenced from increased foraging rates seen for FFA ponds. Availability of catfish also could be increased in the case of disease and poor water quality. Low dissolved oxygen and diseases that infect the gills cause fish to come closer to the surface, near the plane of oxygen diffusion (Stickney 1979). However, the rarity of low dissolved oxygen and poor water quality in our study suggests that these factors may not be important to increasing catfish availability to herons.

In natural habitats, herons concentrate where fish availability is high (Willard 1977, Kushlan 1981). Despite the enormous density of pond-cultured catfish ranging from 2,000 fish/ha at food-fish ponds to 50,000 fish/ha at fingerling ponds (Tucker and Robinson 1990), catfish availability to herons is limited by water turbidity (Secchi disk readings <40 cm at research ponds) and because catfish normally occupy the lower third of the water column (Tucker and Robinson 1990, Glahn et al. 2000). Limited catfish availability also is consistent with findings of previous field studies (Stickley et al. 1995, Glahn et al. 1999b) suggesting that live catfish made up less than half of the heron maintenance diet, estimated at approximately 300 g (Glahn et al. 2000). The remainder of the diet is primarily dead catfish and wild-spawned sunfish. Wild-spawned sunfish have increased availability to visually foraging herons because they spend more time in the littoral zone (Glahn et al. 2000).

Unlike clear-water aquaculture situations (e.g., trout) where fish availability is relatively constant, herons cannot readily meet their daily food demand from typically bottom-dwelling catfish populations (Glahn et al. 2000). Thus, herons at catfish farms have adapted to exploit temporary increases in live catfish availability and the presence of dead catfish and wild-spawned fish (Stickley et al. 1995, Glahn et al. 1999b). This has probably been paramount to their success and expansion in this habitat (Glahn et al. 1999b).

## MANAGEMENT IMPLICATIONS

Great blue herons are perceived to be a widely occurring problem at catfish farms because of the predation damage they inflict (Wywiałowski 1999). Although previous field studies have provided some credence to this notion based solely on bird

numbers and diet (Stickley et al. 1995, Glahn et al. 1999b), they failed to define the circumstances under which predation occurred. Consistent with captive heron studies (Dorr et al. 1998, Glahn et al. 2000), our study demonstrated that herons foraged extensively at ponds with diseased fish and removed mainly fish that were terminally ill. Herons also consume a considerable amount of dead catfish in these situations (Stickley et al. 1995, Glahn et al. 1999b). Although a small percentage of fish taken from these ponds are healthy, the removal of dead and dying fish might help limit the spread of disease to the remaining fish (Waterstrat et al. 1999). In addition, aggregations of herons and other wading birds at ponds may help alert catfish producers to disease problems so they can take remedial actions. Considering that herons prey extensively on diseased fish raises the question of whether they may serve as vectors of fish disease. Herons play little or no role in the transmission of Enteric Septicemia, the most widely occurring disease of farm-raised catfish (Waterstrat et al. 1999). However, herons might serve as vectors of other fish diseases or parasites, particularly when the birds carry infected fish from one pond to another. This being the case, it might be counterproductive to harass herons at infected ponds and disperse them to surrounding ponds.

Heron is inefficient at capturing healthy catfish unless circumstances bring fish to the surface where they are susceptible to predation. The use of floating fish feed briefly brings most of the fish population near the surface and probably results in the most significant loss of healthy catfish to heron predation. However, these losses would appear minor and readily preventable by farm personnel. Because feed is broadcast over ponds from trucks driven along the levee, the simplest procedure would be to have the truck driver or other personnel harass herons with pyrotechnics while feeding ponds. Harassment may have to be carried out only during the summer months, because during the late winter and spring, heron populations are relatively low (Glahn et al. 1999b) and during the fall, herons seem to focus their foraging activity on diseased ponds (this study).

Low dissolved-oxygen levels also can bring healthy catfish to the surface and expose them to heron predation. However, this did not appear to be a problem at the farms we studied because dissolved-oxygen levels were closely monitored and aeration was provided before fish responded by coming to the surface. Thus, good fish manage-

ment practices tend to alleviate predation losses occurring from this circumstance.

Heron predation losses observed in this study may be greater than average because we selected farms reporting significant losses and conducted the study when heron populations were at their highest (Glahn et al. 1999b). Nonetheless, predation losses due to herons in this study appeared negligible. Catfish farmers in the Mississippi Delta region have reported spending an average of US\$4,000/yr to prevent or control predation by herons (Glahn et al. 1999b). Considering the limited nature of heron predation on healthy catfish, such expenditures for control activities may be misplaced and unnecessary. Assuming that heron predation losses observed in this study are similar to those experienced at other catfish farms, we conclude that great blue heron predation at catfish farms is insignificant or readily preventable.

## ACKNOWLEDGMENTS

We thank the Mississippi catfish owners and staff for allowing us to work at their facilities. Discussions with M. E. Tobin contributed significantly to the conceptual development of this study. We also thank G. Ellis of Wildlife Services and P. Gerard with the Mississippi State University Agriculture and Forestry Experiment Station for assistance with data collection and statistical design, respectively. We appreciate helpful comments on earlier manuscript drafts from L. Clark, S. Werner, D. T. King, M. E. Tobin, and M. L. Avery. Heron collections were approved by the Institutional Animal Care and Use Committee of the National Wildlife Research Center and conducted under federal and state scientific collection permits.

## LITERATURE CITED

- BOWSER, P. R., AND J. A. PLUMB. 1980. Growth rates and evaluation of new cell line from channel catfish ovary and channel catfish virus replication at different temperatures. *Canadian Journal of Fisheries and Aquatic Sciences* 37:871–873.
- DORR, B. S., L. CLARK, J. F. GLAHN, AND I. MEZINE. 1998. Evaluation of a methyl anthranilate-based bird repellent: toxicity to channel catfish *Ictalurus punctatus* and effect on great blue heron *Ardea herodias* feeding behavior. *Journal of the World Aquaculture Society* 29:451–462.
- GLAHN, J. F., B. S. DORR, AND M. E. TOBIN. 2000. Captive great blue heron predation on farmed channel catfish fingerlings. *North American Journal of Aquaculture* 62:149–156.
- , J. B. HARREL, AND C. VYLES. 1998. The diet of wintering double-crested cormorants feeding at lakes in the southeastern United States. *Colonial Waterbirds* 21:446–452.



- , E. S. RASMUSSEN, T. TOMSA, AND K. PREUSSER. 1999a. Distribution and relative impact of avian predators at aquaculture facilities in the northeastern United States. *North American Journal of Aquaculture* 61:340–348.
- , D. S. REINHOLD, AND P. SMITH. 1999b. Wading bird depredations on channel catfish *Ictalurus punctatus* in the delta region of Mississippi. *Journal of the World Aquaculture Society* 30:107–114.
- , T. TOMSA, AND K. J. PREUSSER. 1999c. Impact of great blue heron predation at trout-rearing facilities in the northeastern United States. *North American Journal of Aquaculture* 61:349–354.
- HODGES, M. F. 1989. Depredation of channel catfish by birds on Mississippi catfish farms. Thesis, Mississippi State University, Mississippi State, USA.
- HOY, M., J. JONES, AND A. BIVINGS. 1989. Economic impact and control of wading birds at Arkansas minnow ponds. *Proceedings of Eastern Wildlife Damage Control Conference* 4:109–112.
- KUSHLAN, J. A. 1981. Resource use strategies of wading birds. *Wilson Bulletin* 93:145–163.
- LOVELL, R. T. 1989. Nutrition and feeding of fish. Van Nostrand Reinhold, New York, USA.
- PARKHURST, J. A., R. P. BROOKS, AND D. E. ARNOLD. 1992. Assessment of predation at trout hatcheries in central Pennsylvania. *Wildlife Society Bulletin* 20:411–419.
- PITT, W. C., AND M. R. CONOVER. 1996. Predation at intermountain west fish hatcheries. *Journal of Wildlife Management* 60:616–624.
- PLUMB, J. A., AND P. R. BOWSER. 1983. Microbial fish disease laboratory manual. Alabama Agricultural Experiment Station, Montgomery, USA.
- ROSS, P. G. 1994. Foraging ecology of wading birds at commercial aquaculture facilities in Alabama. Thesis, Auburn University, Alabama, USA.
- SAS INSTITUTE. 1994. SAS/STAT user's guide. SAS Institute, Cary, North Carolina, USA.
- . 1996. SAS/STAT changes and enhancements. SAS Institute, Cary, North Carolina, USA.
- STICKLEY, A. R., JR., J. F. GLAHN, J. O. KING, AND D. T. KING. 1995. Impact of great blue heron depredations on channel catfish farms. *Journal of the World Aquaculture Society* 26:194–199.
- STICKNEY, R. R. 1979. Principles of warmwater aquaculture. Wiley-Interscience, New York, USA.
- TUCKER, C. S., AND E. H. ROBINSON. 1990. Channel catfish farming handbook. Chapman & Hall, New York, USA.
- WATERSTRAT, P. R., B. S. DORR, J. F. GLAHN, AND M. E. TOBIN. 1999. Recovery and viability of *Edwardsiella ictaluri* from great blue herons *Ardea herodias* fed *E. ictaluri*-infected channel catfish *Ictalurus punctatus* fingerlings. *Journal of the World Aquaculture Society* 30:115–122.
- WILLARD, D. E. 1977. The feeding ecology and behavior of five species of herons in southeastern New Jersey. *Condor* 79:462–470.
- WYNTALOWSKI, A. P. 1999. Wildlife-caused losses for producers of channel catfish (*Ictalurus punctatus*) in 1996. *Journal of the World Aquaculture Society* 30:461–472.

Received 24 May 2000.

Accepted 14 September 2001.

Associate Editor: Clark.